CASE DEFINITION FOR ABALONE VIRAL MORTALITY (AVM) COMPLEX

General Description

Within the AVM complex, two syndromes have emerged over the past ~15 years: one has an acute course (herpes-like virus disease, HLVD) and the other has a more sub-acute to chronic course (crack-shell-amyotrophia-virus disease, CSAVD). Both syndromes impact multiple abalone species in Australasia (China, Japan, Taiwan, and Australia) with significant losses. However, different clinical courses and presentations lead to currently require different case definitions. Upon comparison of nucleic acid sequences and development of molecular tests, case definitions may change.

Abalone herpes-like virus (AHLV) disease

Known affected species - to date, primarily observed in both subspecies of *Haliotis diversicolor* (*aquatilis* and *supertexta*) and in *Haliotis laevegata, H. rubra* and hybrids of *H. laevegata* x *H. rubra*.

Gross observations - rapid onset of mortality in tanks or ponds with no visible change in abalone feeding habits prior to onset. During outbreaks, tank water is typically turbid and frothy with several reports of suspended, presumably regurgitated, food particles and mucus in water. Affected abalone with clinical signs varying from none to having a stiff pedal muscle with darkened lateral mantle, increased mucus production reported in many cases and may present swollen, prolapsed mouth with everted radula in some cases (noted in Australian abalone species). Mortalities typically observed within 3 days of onset of clinical signs, and dead abalone may remain adhered to substrata. Losses often complete within 9-14d. Losses typically occur when water temperatures are <22C and often range from 16-19C.

Microscopic observations - when used light microscopic observations have suggested that the main pathological change is ganglioneuritis with lesions prominent in cerebral and pedal ganglia[1] . Lesions characterized by nerve tissue necrosis accompanied by hemocytosis in the parenchyma and extend into neurolemma. These lesions can also be observed in nerves under mucosa of esophagus and intestine. No Cowdry type A inclusions were observed; however neuronal cells may contain marginated chromatin.

Transmission electron microscopic (TEM) observations illustrate spherical, enveloped virus (~100nm) with icosahedral (hexagonal) nucleocapid and dense core. Naked virions observed in nucleus and particles with smooth envelop in cytoplasm. Negative-contrast electron microscopy also reveals hexagonal particles with single, smooth envelope (~100nm).

Presumptive diagnosis – a combination of clinical signs and microscopic features as described above.

Confirmatory diagnosis – presumptive diagnosis in conjunction with the presence of spherical virus containing an icosahedral nucleocapsid and dense core using TEM[2]. Occasionally only empty capsids are visible in nucleus of infected cells.

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[1] To date descriptions of the AHLV from China have not included histopathology.

[2] Molecular tests for AHLV are currently under development.

Crack-shell-amyotrophia-virus (CSAV) disease

Known affected species - to date, primarily observed in *Haliotis discus discus* and *H. discus hannai*, and, to a lesser extent, *Haliotis madaka*.

Gross observations – reduced growth and/or abnormal shell deposition, sub-acute or slow losses with up to 50% mortality in 20 days. Affected abalone lethargic with retracted mantle, abnormal shell deposition often posses a thin, cracked shell. Anorexia reported in many cases. Juveniles typically more susceptible than older animals. Water temperature modulates disease with losses often at 18-20C.

Microscopic observations - light microscopic observations suggest the main pathological change in symptomatic animals includes the presence of tumor-like masses presented as whorls or spheres of lightly basophilic cells within nerve trunks of pedal ganglia and transverse commissures ('gliomas'). Nuclei of affected cells may be contracted and tumor centers necrotic[3].

Transmission electron microscopic (TEM) observation may reveal 90-140nm spherical, enveloped virions with an icosahedral nucleocapsid in cells near nerves and in the cytoplasm of hemocytes and connective tissue cells of a variety of organs.

Presumptive diagnosis – a combination of clinical signs and microscopic features as described above.

Confirmatory diagnosis – presumptive diagnosis in conjunction with the presence of 90-140nm spherical, enveloped virions with an icosahedral nucleocapsid in infected cells.

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[3] To date descriptions of the CSAV from China have not included histopathology